

# Prevalence of *Campylobacter jejuni* and *Campylobacter coli* in Market-Weight Turkeys On-Farm and at Slaughter†

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## ABSTRACT

To monitor the effects of feed withdrawal on the prevalence of *Campylobacter*, market-weight turkeys from six farms were examined before and after perimarketing events (feed withdrawal, transport, and holding at the slaughterhouse). Prior to transport, birds ( $n = 30$  per farm) were slaughtered on-farm, and viscera (crops, duodenum, jejunum, ileum, colon, ceca, gallbladder, and spleen) were removed on the premises. Within ca. 48 h, cohorts ( $n = 30$  per farm) from the same flock were transported to a commercial abattoir, maintained in holding sheds, slaughtered, and the viscera were removed. No differences in the prevalence of *Campylobacter* spp. were evident when individual flocks were compared pre- and posttransport. However, when data for the six farms were combined, *Campylobacter* spp. were recovered (pre- versus posttransport) at comparable rates from the duodenum (74.7 versus 74.7%), ileum (87.3 versus 92.7%), ceca (64 versus 57%), colon (86.7 versus 80%), and spleen (0 versus 0%). After feed withdrawal, transport, and holding at the abattoir, there was an overall increase in *Campylobacter* spp. isolated from the gallbladder at the abattoir (14.7%) when compared with on-farm levels (0%,  $P < 0.05$ ). When compared with on-farm levels (3%), the overall increase in *Campylobacter* spp. recovered from the crops of birds at the abattoir (24%) was significant ( $P < 0.05$ ), which may be associated with a detectable decline in lactic acid in the emptied crop.

In the United States, the nearly 2 million cases of human foodborne campylobacteriosis result in ca. 10,000 hospitalizations and ca. 100 deaths annually (20). Consumption of contaminated, undercooked poultry is a major risk factor for human *Campylobacter* infections (16, 22). The 1997 U.S. Department of Agriculture, Food Safety and Inspection Service young turkey baseline study detected *Campylobacter jejuni* and *Campylobacter coli* on 90% of turkey carcasses ([www.fsis.usda.gov/ophs/baseline/yngturk1.pdf](http://www.fsis.usda.gov/ophs/baseline/yngturk1.pdf)). The observed decline in human campylobacteriosis from 1996 to 1999 coincided with the implementation of hazard analysis and critical control point testing in poultry processing plants (27). Since mathematical models predict that reducing *Campylobacter* in poultry meat will impact public health, reducing the prevalence of *Campylobacter* spp. in live turkeys entering the abattoir may decrease human campylobacteriosis (16, 22, 29).

Perimarketing events, including feed withdrawal, catching, crating, and transport to the abattoir within ca. 24 h of slaughter, may influence intestinal carriage of *Campylobacter* spp. in broilers and thus the extent of fecal carcass contamination (8, 19, 32, 35). Whyte et al. (38) concluded that although the incidence was unchanged, the number of *Campylobacter* spp. in feces of broilers in-

creased significantly after transportation. In addition, contact with soiled crates may contaminate birds after leaving the farm, en route to the abattoir (13, 23, 30).

In studies of commercial turkey flocks to determine if marketing stresses also influenced *Salmonella* prevalence, no differences were observed when individual flocks were analyzed before and after feed withdrawal, transport, and holding at a commercial abattoir (25, 36). However, when data were combined for the six turkey flocks, a significant increase was observed in *Salmonella* isolated from liver and gallbladder tissue, which the authors speculated was attributed to neuroendocrine changes associated with perimarketing events (25).

*Salmonella* and *Campylobacter* colonize the avian crop, which is a potential source of carcass contamination at slaughter. The prevalence of *Campylobacter* and *Salmonella* in the broiler crop increase with feed withdrawal (4, 10); thus, preslaughter treatments, such as lactic acid, have been evaluated for their ability to reduce *Salmonella* and *Campylobacter* in broilers (5).

Few studies detail the effect of perimarketing events on the prevalence of *Campylobacter* spp. in commercial turkey flocks when feed withdrawal, transport to, and holding at the abattoir occur (37). Therefore, the primary goal of this study was to determine if perimarketing events influence the prevalence of *Campylobacter* spp. in viscera of market-weight turkeys. To this end, the prevalence of *Campylobacter* spp. recovered from turkeys slaughtered on-farm was compared with that of cohorts transported to and slaughtered at a single commercial abattoir.

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† Mention of trade names or commercial products in this publication is solely for the purpose of providing specific information and does not imply recommendation or endorsement by the U.S. Department of Agriculture.

TABLE 1. Summary of management practices for each of the farms in this study

Parameter	Farm no.:					
	1	2	3	4	5	6
Turkey						
Hatchery	A	B	A/B	B/D	B	C
Poultry strain	Hybrid	Hybrid	Hybrid	Hybrid	Hybrid	Hybrid
Growing program	Two stage	Three stage	Two stage	Two stage	Two stage	Three stage
Feed						
Feed mill	D	E	E	E	E	E
Growth antibiotics	Virginiamycin	Virginiamycin	Bacitracin, virginiamycin	Flavomycin	Virginiamycin	Virginiamycin
Level (g/ton)	20	10			10–20	20
Age fed	9 wk to market	15 lb to market wt	Unknown	9 wk to market	Unknown	Unknown
Water						
Water source	Farm well	Farm well	Farm well	Farm well	Farm well	Farm well
Chlorination	No	Yes	Yes	Yes	Yes	Peroxide
Last clean water lines	Unknown	Every 2 months	No	Prior to this flock	Each flock	Every 2 mo
Water treatment preload	Unknown	Electrolytes	No	No	No	No
Litter						
Litter type	Wood shavings	Sawdust	Oat hulls, sawdust	Oat hulls, sawdust	Oat hulls, sawdust	Oat hulls, sawdust
Last changed	2 yr ago, scrape and top dress	2 yr ago, dress prior to flock	5 yr ago	Prior to this flock	Top dress, after cake removed	1 yr ago
Treatment	Unknown	F	G	H	G	None
Animals						
Livestock	No	No	Cattle	No	No	No
Animals in house	Yes (dogs)	No	No	No	No	No
Mice/rats	Mice	No	Mice	No	No	Yes
Birds (sparrows)	Yes	No	Yes	No	Seldom	Yes
Flock health						
Vaccines used	None	HE <sup>a</sup>	HE, NDV <sup>b</sup>	HE, NDV	HE, NDV	None
Diseases	Crop mycosis	Pneumovirus	Pneumovirus	No	No	No
Antibiotic treatment	Yes	Yes	Yes	Yes	No	No
Coccidiostat used	Yes	Yes	Yes	Yes	Yes	Yes
Biosecurity						
Foot bath required	No	Yes	Yes	Yes	Yes	Yes
Disinfectant	N/A	Yes	Yes	Yes	No	Yes
Boot change	No	Yes	Yes	No	No	Yes
Clothes change	No	Yes	Yes	No	No	No

<sup>a</sup> HE, hemorrhagic enteritis.  
<sup>b</sup> NDV, Newcastle disease virus.

MATERIALS AND METHODS

**Farm selection.** Six Midwestern turkey premises were selected based on previous analysis of these flocks and owners’ cooperation. The farms utilized commercial production systems as described in Table 1, and shipped turkeys to a single commercial abattoir that processed ca. 20,000 birds daily during the summer months when this study was conducted.

**Duration of perimarketing events.** The total of time off-feed encompasses the interval from the time birds are loaded onto transport vehicles to the time birds are shackled. This includes transport and resting (lairage) in the holding shed. The total time off-feed for each of the six premises was based on drivers’ logs

and the records of the slaughter plant. To minimize the stress of perimarketing events and cross contamination (13), turkeys sampled were the first group to be vacated from the house.

**Viscera sampling pre- and posttransport.** On each farm premise within ca. 48 h before depopulation, market-weight turkeys (*n* = 30 per farm) were randomly selected, humanely euthanized as outlined in Iowa State University Institution Care and Use Committee protocol 8-03-5512-T, and viscera (crops, duodenum, jejunum, ileum, colon, ceca, gallbladder, and spleen) were aseptically removed, as described in (25). Gallbladder and surrounding liver tissue were removed and processed as gallbladder-liver. Within ca. 48 h of on-farm sampling, the flock was trans-

TABLE 2. Summary of times off-feed for turkeys on each of the six premises<sup>a</sup>

Farm no.	Feeders lifted pre-load out (h)	Loading time (h)	Transit time (h)	Holding time (h)	Time off-feed (h)
1	Unknown	0.82	3.03	3.82	7.67
2	3	0.86	3.40	4.13	11.41
3	4	0.78	0.67	8.62	14.07
4	4	0.74	2.89	3.17	10.80
5	2	0.9	2.77	5.48	11.15
6	1	0.80	2.91	3.61	8.96
Mean <sup>b</sup>	2.8 ± 1.3	0.82 ± 0.1	2.61 ± 0.97	4.80 ± 2.02	10.68 ± 2.20
Range	1–4	0.74–0.9	0.67–3.4	3.17–8.62	7.67–14.07

<sup>a</sup> Time off-feed includes time feeders are lifted as well as times for loading, transport, and lairage in the holding shed.  
<sup>b</sup> Values are means ± standard deviations.

ported to a commercial abattoir, maintained in holding sheds, and slaughtered. At the evisceration point, viscera were removed from cohorts ( $n = 30$  per farm) by using sterile forceps and scissors for each organ. At both pre- and posttransport sampling times, organs were placed in Whirl-Pak bags (Nasco, Ft. Atkins, WI), transported to the laboratory on ice, and processed the same day.

**Campylobacter spp. isolation.** Organs were processed for *Campylobacter* isolation, and presumptive *Campylobacter* colonies were identified as *C. jejuni* or *C. coli* by using PCR primers, as described by Wesley et al. (37).

**Carboxylic acid analysis.** Gas chromatography of butyl esters was used to determine the concentration of organic acids (expressed in micromolar concentrations) for one randomly selected flock, as described by Cutler et al. (9). Crop samples were analyzed pretransport ( $n = 30$ ) and posttransport ( $n = 20$ ). Cecal samples were evaluated pretransport ( $n = 30$ ) and posttransport ( $n = 30$ ).

**Statistical analysis.** Paired Student's  $t$  tests were used to compare the significant differences in the mean prevalence of *Campylobacter* spp., *C. jejuni*, and *C. coli* in turkeys slaughtered on-farm with those slaughtered at the commercial abattoir. A value of  $P < 0.05$  was regarded as significant. The overall prevalence of *Campylobacter* spp., *C. jejuni*, and *C. coli*, which combined data for the six flocks, was compared for all organs. Differences between pre- and posttransport values for carboxylic acids were analyzed using appropriate equal or unequal variance Student's  $t$

tests, separately for both crop and ceca specimens. All analyses were done using SAS for PC Windows, version 9.1.3.

RESULTS

Times (means ± standard deviations, expressed in hours) feeders are lifted prior to load out, to load at the farm ( $0.82 \pm 0.06$  h), for transit to the slaughterhouse ( $2.61 \pm 0.97$  h), and for resting at the slaughterhouse ( $4.8 \pm 2.02$  h) are summarized in Table 2. The estimated time off-feed for the six flocks was  $10.68 \pm 2.2$  h.

No differences were noted in prevalence when individual flocks were compared for *Campylobacter* spp., *C. jejuni*, or *C. coli* pre- and posttransport. However, as summarized in Table 3, when prevalence data for the six farms are combined, for turkeys slaughtered on-farm, *Campylobacter* spp. representing *C. jejuni* and *C. coli* combined were recovered from crop (3%), duodenum (74.7%), ileum (87.3%), ceca (64%), and colon (86.7%). *Campylobacter* spp. were not isolated from either the gallbladder or spleen. After feed withdrawal, transport, and holding at the abattoir, *Campylobacter* spp. were recovered from the crop (24%), duodenum (74.7%), ileum (92.7%), ceca (57%), and colon (80%). The *Campylobacter* spp. prevalence in the crops of birds slaughtered after transport (24%) was significantly higher ( $P < 0.05$ ) than that for turkeys slaughtered on-farm

TABLE 3. Isolation of *Campylobacter* spp. from the viscera of market-weight turkeys<sup>a</sup>

Transport level	Organ:						
	Crop ( $n = 180$ )	Duodenum ( $n = 150$ )	Gallbladder ( $n = 150$ )	Spleen ( $n = 180$ )	Ileum ( $n = 150$ )	Ceca ( $n = 180$ )	Colon ( $n = 180$ )
Pretransport							
<i>Campylobacter</i> spp.	3 (6)	74.7 (112)	0 (0)	0 (0)	87.3 (131)	64 (115)	86.7 (156)
<i>C. coli</i>	1.2 (2)	10.7 (16)	0 (0)	0 (0)	18.7 (28)	43.9 (79)	23.3 (42)
<i>C. jejuni</i>	2.2 (4)	72 (108)	0 (0)	0 (0)	84.7 (127)	27.8 (50)	78.9 (142)
Posttransport							
<i>Campylobacter</i> spp.	24 <sup>b</sup> (43)	74.7 (112)	14.7 <sup>b</sup> (22)	2.2 (4)	92.7 (139)	57 (103)	80 (144)
<i>C. coli</i>	13.9 (25)	14.7 (22)	4 (6)	0 (0)	42 (63)	40 (72)	41 (74)
<i>C. jejuni</i>	10.6 (19)	67.3 (101)	11.3 <sup>b</sup> (17)	2.2 (4)	66.7 (100)	24.4 (44)	55.6 (100)

<sup>a</sup> Data are from six flocks slaughtered on-farm ( $n = 30$ ) and at the abattoir ( $n = 30$ ), are shown as percentage (number) positive per organ, and are a composite of six flocks.  
<sup>b</sup> Represents a significant increase ( $P < 0.05$ ) in *Campylobacter* after transport.

TABLE 4. Summary of carboxylic acids in crops<sup>a</sup> pre- and post-transport

Acid	Pretransport ( $\mu\text{M} \pm \text{SEM}$ )	Posttransport ( $\mu\text{M} \pm \text{SEM}$ )
Formic	5.46 $\pm$ 2.6	0.37 $\pm$ 0.09
Acetic <sup>b</sup>	3.84 $\pm$ 0.42	2.4 $\pm$ 0.41
Propionic	0.16 $\pm$ 0.04	0.49 $\pm$ 0.22
Isobutyric <sup>b</sup>	0.06 $\pm$ 0.01	0.02 $\pm$ 0.00
Butyric	0.09 $\pm$ 0.03	0.05 $\pm$ 0.02
Lactic <sup>b</sup>	24.84 $\pm$ 1.61	13.25 $\pm$ 3.14
Isovaleric	0.14 $\pm$ 0.04	0.06 $\pm$ 0.03
Valeric <sup>c</sup>	0.021 $\pm$ 0.01	0.11 $\pm$ 0.01
Caproic <sup>b</sup>	0.10 $\pm$ 0.03	0.02 $\pm$ 0.01
Oxalic <sup>b</sup>	0.38 $\pm$ 0.13	0.11 $\pm$ 0.04
Phenyl acetic	0.67 $\pm$ 0.61	0.09 $\pm$ 0.01
Succinic	1.17 $\pm$ 0.12	1.14 $\pm$ 0.16
Fumaric	0.01 $\pm$ 0.01	0 $\pm$ 0
Total	31.31 + 2.25	17.72 + 3.48

<sup>a</sup> *n* = 30 pretransport; *n* = 20 posttransport.  
<sup>b</sup> Represents a significant reduction (*P* < 0.05) after transport.  
<sup>c</sup> Represents a significant increase (*P* < 0.05) after transport.

(3%). Likewise, the *Campylobacter* spp. prevalence in gallbladder at the abattoir (14.7%) when compared to on-farm pretransport levels (0%) was significantly higher (*P* < 0.05). No such differences were noted for *Campylobacter* spp. isolated from the duodenum, ileum, ceca, colon, or spleen.

When the prevalence of *C. coli* was combined for all six premises, marginally more *C. coli* were recovered from the crop posttransport (13.9%) when compared with pretransport levels (1.2%, *P* < 0.07). No differences were noted for *C. coli* in other organs.

When the prevalence of *C. jejuni* was combined for all six premises, significantly more *C. jejuni* were recovered from the gallbladder posttransport (11.3%) when compared with pretransport levels (0%, *P* < 0.05, Table 3). No differences were noted for *C. jejuni* in the other organs.

Carboxylic acids (means  $\pm$  standard errors of the means, expressed in micromolar concentrations) in the crop and ceca pre- and posttransport were evaluated for one flock. For the crop, as seen in Table 4, the concentrations of total organic acids (31.31  $\pm$  2.25  $\mu\text{M}$ ) and lactic acid in bird slaughtered on farm pretransport (24.84  $\pm$  1.61  $\mu\text{M}$ ) were significantly higher (*P* < 0.05%) than those slaughtered posttransport (17.72  $\pm$  3.48 and 13.25  $\pm$  3.14  $\mu\text{M}$ , respectively). After time off-feed posttransport, a significant decrease (*P* < 0.05%) in acetic, isobutyric, caproic, and oxalic acids was noted; valeric acid concentrations increased posttransport (*P* < 0.05%). For the ceca, as seen in Table 5, after time off-feed posttransport, a significant increase in formic, isobutyric, isovaleric, valeric, and oxalic acids was noted. Lactic acid concentrations in the ceca at both pre- (0.41  $\pm$  0.33  $\mu\text{M}$ ) and posttransport (0.12  $\pm$  0.038  $\mu\text{M}$ ) were lower than those in the crop of either the fed (24.84  $\pm$  1.61  $\mu\text{M}$ ) or asted (13.25  $\pm$  3.14  $\mu\text{M}$ ) birds.

TABLE 5. Summary of carboxylic acids in ceca<sup>a</sup> pre- and post-transport

Acid	Pretransport ( $\mu\text{M} \pm \text{SEM}$ )	Posttransport ( $\mu\text{M} \pm \text{SEM}$ )
Formic <sup>b</sup>	0.37 $\pm$ 0.03	0.61 $\pm$ 0.03
Acetic	25.62 $\pm$ 1.91	27.3 $\pm$ 1.22
Propionic	9.19 $\pm$ 0.63	8.35 $\pm$ 0.43
Isobutyric <sup>b</sup>	0.61 $\pm$ 0.06	0.99 $\pm$ 0.06
Butyric	6.75 $\pm$ 0.60	5.5 $\pm$ 0.39
Lactic	0.41 $\pm$ 0.33	0.12 $\pm$ 0.04
Isovaleric <sup>b</sup>	0.80 $\pm$ 0.09	1.11 $\pm$ 0.06
Valeric <sup>c</sup>	0.76 $\pm$ 0.10	1.23 $\pm$ 0.07
Oxalic <sup>b</sup>	0.25 $\pm$ 0.03	0.34 $\pm$ 0.02
Caproic	0.02 $\pm$ 0.01	0.01 $\pm$ 0.01
Phenyl acetic	0.18 $\pm$ 0.03	0.23 $\pm$ 0.02
Succinic	0.48 $\pm$ 0.04	0.49 $\pm$ 0.03
Fumaric	0.16 $\pm$ 0.05	0.19 $\pm$ 0.04
Total	44.89 + 2.95	46.6 + 2.20

<sup>a</sup> *n* = 30 pretransport; *n* = 30 posttransport.  
<sup>b</sup> Represents a significant reduction (*P* < 0.05) after transport.  
<sup>c</sup> Represents a significant increase (*P* < 0.05) after transport.

DISCUSSION

The primary goal of this study was to compare the prevalence of *Campylobacter* spp. in viscera of normally fed, commercially reared market-weight turkeys killed on-farm with those of fasted cohorts after commercial live haul, resting, and slaughter at the abattoir. To minimize differences in farm management practices, we sampled birds from premises that belonged to members of a growers' co-operative and utilized the same live haul company for transport to the single abattoir.

In this study, the distribution of *Campylobacter* spp. along the intestinal tract was examined. When the six individual flocks were compared, there were no statistically significant differences in recovery rates of *Campylobacter* spp., *C. jejuni*, or *C. coli* pre- and posttransport. When combined data for the flocks are analyzed (all turkeys slaughtered on-farm versus all turkeys slaughtered at the abattoir), significantly more *Campylobacter* spp. were isolated after fasting from crop (3 versus 24%, *P* < 0.05%). This difference may reflect changes in organic acids in the empty crop as well as the decreased dilution with feed.

*Campylobacter* and *Salmonella* colonize the chicken crop (with prevalence elevated after fasting), which may be a potential source of carcass contamination at slaughter (4, 10, 24). Consequently, lactic, propionic, and formic, and other organic acids incorporated into the drinking water have been evaluated for their ability to reduce foodborne pathogens in the crop or ceca of broilers and to mitigate subsequent carcass contamination with bacterial foodborne pathogens (1, 5, 6, 34). Although comparison of broiler and turkey processing is beyond the scope of this report, overall turkey processing is more labor-intensive when compared with broiler processing. Specifically, whereas for turkeys, evisceration—including removal of the crop and ceca—is manually performed, it is fully automated during broiler processing.



Few reports have correlated the biochemical changes associated with fasting or diminished feed uptake in the turkey crop with susceptibility to foodborne bacterial pathogens (9, 14). In contrast, in our studies with a single commercial turkey flock off-feed ca. 11 h prior to slaughter, the reduction for lactic acid most noticeably coincided with an increase in *Campylobacter* spp. in the crop. This concurs with the observed efficacy of lactic acid-supplemented drinking water as a pathogen-reduction intervention prior to slaughter (5) at levels exceeding its function as a significant electron donor in *Campylobacter*. Interestingly, our companion study evaluating these same birds, utilizing commercial transport and holding conditions, showed no change in *Salmonella* in the crop (25). Feed intake naturally declines at night when birds enter the scotophase. The observed decline in lactic acid in commercial adult turkeys contrasts with the increase seen in week-old turkey poults at 8 h during the experimentally induced scotophase (9).

When data for the six flocks are combined, the observed increase in *Campylobacter* spp. isolated from the gallbladder-liver of turkeys slaughtered after transport when compared with cohorts necropsied on the farm may reflect accumulation of bile during fasting and the subsequent enlargement of the gallbladder. Expression of virulence genes of *Campylobacter* is stimulated in vitro with the bile salt deoxycholate (17, 18), which suggests that the gallbladder is a favorable habitat. *Campylobacter* is infrequently isolated from poultry gallbladder (3, 40). In contrast, *Campylobacter* has been isolated from the bile of cattle and as such, presents a risk of contamination of beef liver (11, 26). *Campylobacter* has been isolated from chicken but not turkey livers, and one outbreak in humans incriminated chicken liver pâté as the vehicle of transmission (26, 39).

Our earlier study utilizing these same birds found no significant change in *Salmonella* in any of the viscera examined when individual flocks were compared pre- and posttransport. However, when data for the six premises were combined, significantly more *Salmonella* was isolated from the gallbladder and liver (25). We speculated that this could be attributed to neuroendocrine changes associated with perimarketing events, since norepinephrine enhances the virulence and motility of *Salmonella* in cattle and pigs and increases the virulence of *Campylobacter* in vitro (2, 7).

We hypothesized the presence of *Campylobacter* in the spleen would reflect bacteremia as a result of perimarketing stress. While blood collection is achievable on-farm, commercial line processing speeds preclude sampling at the abattoir. Thus, the spleen, the single largest lymphatic organ of turkeys, was selected for evaluation. However, *Campylobacter* spp. were not cultured from the spleens of adult turkeys at either sampling interval, suggesting no bacteremia associated with perimarketing events. No reports detailing the prevalence of *Campylobacter* in the spleens of adult turkeys are available for comparison.

In the northern hemisphere, *C. jejuni* is the major cause of bacterial human gastroenteritis. To illustrate, FoodNet sites identified laboratory-confirmed *Campylobacter* isolates as *C. jejuni* (95%), *C. coli* (4%), and *C. lari* (1%)

(12). However, *C. coli* is gaining recognition as a zoonotic foodborne pathogen, and in the United Kingdom, *C. coli* ranks fourth as a cause of bacterial foodborne illness (33). Although regarded predominantly as an intestinal commensal of swine, *C. coli* is commonly isolated from turkeys (15, 21, 31, 37). In addition, Siemer et al. (28) estimated that 9 to 21% of *Campylobacter* poultry and clinical isolates identified as *C. jejuni* were in fact *C. coli*, suggesting that the lower prevalence of *C. coli* may result from their misidentification.

In conclusion, we have shown that the overall prevalence of *Campylobacter* spp. increases in the crop and gallbladder of turkeys during the perimarketing interval, under commercial conditions. Whether the observed changes result from fluctuations in organic acids in the crop, a neuroendocrine surge or other factors is not known.

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## REFERENCES

1. Al-Tarzi, Y. H., and K. Alshawabkeh. 2003. Effect of dietary formic and propionic acids on *Salmonella pullorum* shedding and mortality in layer chicks after experimental infection. *J. Vet. Med. B Infect. Dis. Vet. Public Health* 50:112–117.
2. Bearson, B. L., and S. M. Bearson. 2008. The role of the QseC quorum-sensing sensor kinase in colonization and norepinephrine-enhanced motility of *Salmonella enterica* serovar Typhimurium. *Microb. Pathog.* 44:271–278.
3. Beery, J. T., M. B. Hugdahl, and M. P. Doyle. 1988. Colonization of gastrointestinal tracts of chicks by *Campylobacter jejuni*. *J. Appl. Environ. Microbiol.* 54:2365–2370.
4. Byrd, J. A., D. E. Corrier, M. E. Hume, R. H. Bailey, L. H. Stanker, and B. M. Hargis. 1998. Effect of feed withdrawal on *Campylobacter* in the crops of market-age broiler chickens. *Avian Dis.* 42:802–806.
5. Byrd, J. A., B. M. Hargis, D. J. Caldwell, R. H. Bailey, K. L. Herron, J. L. McReynolds, R. L. Brewer, R. C. Anderson, K. M. Bischoff, T. R. Callaway, and L. F. Kubena. 2001. Effect of lactic acid administration in the drinking water during pre-slaughter feed withdrawal on *Salmonella* and *Campylobacter* contamination of broilers. *Poult. Sci.* 80:278–283.
6. Chaveerach, P., D. A. Keuzenkamp, L. J. Lipman, and F. Van Knapen. 2004. Effect of organic acids in drinking water for young broilers on *Campylobacter* infection, volatile fatty acid production, gut microflora and histological cell changes. *Poult. Sci.* 83:330–334.
7. Cogan, T. A., A. O. Thomas, L. E. Rees, A. H. Taylor, M. A. Jepson, P. H. Williams, J. Kettle, and T. J. Humphrey. 2007. Norepinephrine increases the pathogenic potential of *Campylobacter jejuni*. *Gut* 56:1060–1065.
8. Corrier, D. E., J. A. Byrd, B. M. Hargis, M. E. Hume, R. H. Bailey, and L. H. Stanker. 1999. Presence of *Salmonella* in the crop and

- ceca of broiler chickens before and after preslaughter feed withdrawal. *Poult. Sci.* 78:45–49.
9. Cutler, S. A., M. A. Rasmussen, M. J. H. Hensley, K. W. Wilhelms, R. W. Griffith, and C. F. Scanes. 2005. Effects of *Lactobacilli* and lactose on *Salmonella typhimurium* colonization and microbial fermentation in the crop of the young turkey. *Br. Poult. Sci.* 46:708–716.
  10. Durant, J. A., D. E. Corrier, J. A. Byrd, L. H. Stanker, and S. C. Ricke. 1999. Feed deprivation affects crop environment and modulates *Salmonella enteritidis* colonization and invasion of leghorn hens. *Appl. Environ. Microbiol.* 65:1919–1923.
  11. Enokimoto, M., M. Kubo, Y. Bozono, Y. Mieno, and N. Misawa. 2007. Enumeration and identification of *Campylobacter* species in the liver and bile of slaughtered cattle. *Int. J. Food Microbiol.* 188: 259–263.
  12. Friedman, C. R., R. M. Hoekstra, M. Samuel, R. Marcus, J. Bender, B. Shiferaw, S. Reddy, S. D. Ahuja, D. L. Helfrick, F. Hardnett, M. Carter, B. Anderson, R. V. Tauxe, and the Emerging Infections Program FoodNet Working Group. 2004. Risk factors for sporadic *Campylobacter* infection in the United States: a case-control study in FoodNet sites. *Clin. Infect. Dis.* 38(Suppl.):S285–S296.
  13. Hansson, I., M. Ederoth, L. Andersson, I. Vagsholm, and E. Olsson Engvall. 2005. Transmission of *Campylobacter* spp. to chickens during transport to slaughter. *J. Appl. Microbiol.* 99:1149–1157.
  14. Johannsen, S. A., R. W. Griffith, I. V. Wesley, and C. G. Scanes. 2004. *Salmonella enterica* serovar Typhimurium colonization of the crop in the domestic turkey: influence of probiotic and prebiotic treatment (*Lactobacillus acidophilus* and lactose). *Avian Dis.* 48: 279–286.
  15. Lee, B. C., N. Reimers, H. J. Barnes, C. D. Lima, D. Carver, and S. Kathariou. 2005. Strain persistence and fluctuation of multiple-antibiotic-resistant *Campylobacter coli* colonizing turkeys over successive production cycles. *Foodborne Pathog. Dis.* 2:103–110.
  16. Lee, M. D., and D. G. Newell. 2006. *Campylobacter* in poultry: filling an ecological niche. *Avian Dis.* 50:10–9.
  17. Lin, J., C. Cagliero, B. Guo, Y. W. Barton, M. C. Maurel, S. Payot, and Q. Zhang. 2005. Bile salts modulate expression of the CmeABC multidrug efflux pump in *Campylobacter jejuni*. *J. Bacteriol.* 187: 7417–7424.
  18. Malik-Kale, P., C. T. Parker, and M. E. Konkel. 2008. Culture of *Campylobacter jejuni* with sodium deoxycholate induces virulence gene expression. *J. Bacteriol.* 190:2286–2297.
  19. May, J. D., and J. W. Deaton. 1989. Digestive tract clearance of broilers cooped or deprived of water. *Poult. Digest* 68:627–630.
  20. Mead, P. S., L. Slutsker, V. Dietz, L. F. McCaig, J. S. Breesee, C. Shapiro, M. Griffin, and R. V. Tauxe. 1999. Food-related illness and death in the United States. *Emerg. Infect. Dis.* 5:607–625.
  21. Miller, W. G., M. D. Englen, S. Kathariou, I. V. Wesley, G. Wang, L. Pittenger-Alley, R. M. Siletz, W. Muraoka, P. Fedorka-Cray, and R. E. Mandrell. 2005. Identification of host-specific alleles by multilocus sequence typing (MLST) of *Campylobacter coli* isolates from food animals. *Microbiology* 152:245–255.
  22. Nauta, M. J., W. F. G. Jacobs-Reitsma, and A. H. Havelaar. 2007. A risk assessment model for *Campylobacter* in broiler meat. *Risk Anal.* 27:845–861.
  23. Newell, D. G., J. E. Shreeve, T. Toszeghy, G. Domingue, S. Bull, T. Humphrey, and G. Mead. 2001. Changes in the carriage of *Campylobacter* strains by poultry carcasses during processing in abattoirs. *Appl. Environ. Microbiol.* 67:2636–2640.
  24. Rasschaert, G., K. Houf, J. Van Hende, and L. De Zutter. 2006. *Campylobacter* contamination during poultry slaughter in Belgium. *J. Food Prot.* 69:27–33.
  25. Rostagno, M. H., I. V. Wesley, D. W. Trampel, and H. S. Hurd. 2006. *Salmonella* prevalence in market-age turkeys on-farm and at slaughter. *Poult. Sci.* 85:1838–1842.
  26. Saito, S., J. Yatsuyanagi, S. Harata, Y. Ito, K. Shinagawa, N. Suzuki, K. Amano, and K. Enomoto. 2005. *Campylobacter jejuni* isolated from retail poultry meat, bovine feces and bile, and human diarrheal samples in Japan: comparison of serotypes and genotypes. *FEMS Immunol. Med. Microbiol.* 4:311–319.
  27. Samuel, M. C., D. J. Vugia, S. Shallow, R. Marcus, S. Segler, T. McGovern, H. Kassenborg, K. Reilly, M. Kennedy, F. Angulo, and R. V. Tauxe. 2004. Epidemiology of sporadic *Campylobacter* infection in the United States and declining trend in incidence, FoodNet 1996–1999. *Clin. Infect. Dis.* 38(Suppl.):S165–S174.
  28. Siemer, B. L., E. M. Nielsen, and S. L. W. On. 2005. Identification and molecular epidemiology of *Campylobacter coli* isolates from human gastroenteritis, food, and animal sources by amplified fragment length polymorphism analysis and Penner serotyping. *Appl. Environ. Microbiol.* 71:1953–1958.
  29. Singer, R. S., L. A. Cox, J. S. Dickson, H. S. Hurd, I. Phillips, and G. Y. Miller. 2007. Modeling the relationship between food animal health and human foodborne illness. *Prev. Vet. Med.* 79:186–203.
  30. Slader, J., G. Dominique, F. Jorgensen, K. McAlpine, R. J. Owen, F. J. Bolton, and T. J. Humphrey. 2002. Impact of transport crate reuse and of catching and processing on *Campylobacter* and *Salmonella* contamination of broiler chickens. *Appl. Environ. Microbiol.* 68: 713–719.
  31. Smith, K., N. Reimeers, H. J. Barnes, B. C. Lewke, R. Siletzky, and S. Kathariou. 2004. *Campylobacter* colonization of sibling turkey flocks reared under different management conditions. *J. Food Prot.* 67:1463–1468.
  32. Stern, N. J., M. R. S. Clavero, J. S. Bailey, N. A. Cox, and M. C. Robach. 1995. *Campylobacter* spp. in broilers on the farm and after transport. *Poult. Sci.* 74:937–941.
  33. Tam, C. C., S. J. P. O'Brien, G. K. Adak, S. M. Meakins, and J. A. Frost. 2003. *Campylobacter coli*—an important foodborne pathogen. *J. Infect.* 47:28–32.
  34. Thompson, J. L., and M. Hinton. 1997. Antibacterial activity of formic and propionic acids in the diet of hens on salmonellas in the crop. *Br. Poult. Sci.* 38:59–65.
  35. Wabek, C. J. 1972. Feed and water withdrawal time relationship to processing yield and potential fecal contamination of broilers. *Poult. Sci.* 51:1119–1121.
  36. Wesley, I. V., E. Harbaugh, D. Trampel, F. Rivera, and H. S. Hurd. 2005. The effect of perimarketing events on the prevalence of *Salmonella* in market-weight turkeys. *J. Food Prot.* 69:1785–1793.
  37. Wesley, I. V., W. T. Muraoka, D. Trampel, and H. S. Hurd. 2005. The effect of perimarketing events on the prevalence of *Campylobacter jejuni* and *Campylobacter coli* in market-weight turkeys. *Appl. Environ. Microbiol.* 71:2824–2831.
  38. Whyte, P., J. D. Collins, K. McGill, C. Monahan, and H. O'Mahony. 2001. The effect of transportation stress on excretion rates of campylobacters in market-age broilers. *Poult. Sci.* 80:817–820.
  39. Whyte, R., J. A. Hudson, and C. Graham. 2006. *Campylobacter* in chicken livers and their destruction by pan frying. *Lett. Appl. Microbiol.* 43:591–595.
  40. Yusufu, H. I., C. Genigeorgis, T. B. Farver, and J. M. Wempe. 1983. Prevalence of *Campylobacter jejuni* at different sampling sites in two California turkey processing plants. *J. Food Prot.* 46:868–872.